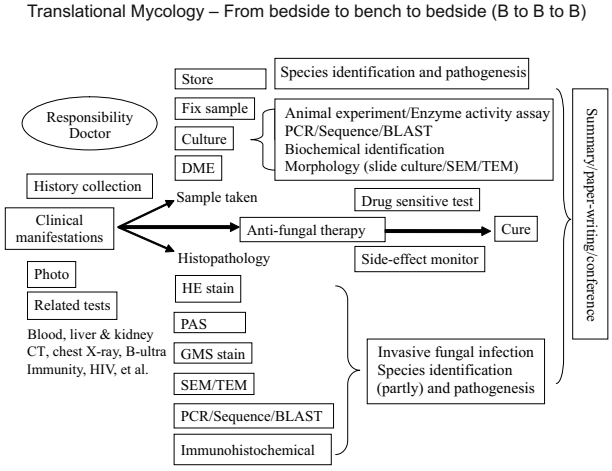
clinical (bedside) and laboratory (bench). The clinician always wants to know what the fungus is and how to treatment the mycosis. Fungal pathogens are often stealthy and difficult to detect in infected patients during the early stages of the diseases and this is when therapies would be the most effective. Routine techniques commonly employed in the detection of fungal diseases including microscopic examination, culturing and serology are seriously hampered by lengthy waits of times for results and low accuracy. The clinician may want prophylaxis or to use empirical antifungal treatment to see if it does/does not work. The problem is that some of the patients do not respond to the antifungal treatment, because the doctor lacked sufficient evidence of fungus infection to give the doctor confidence to continue treatment. Accurate and early diagnosis of fungal diseases is critical for managing mycotic diseases. Our experience is before starting antifungal treatment, we need to be sure the tissue was invaded by a fungus. This is usually done by direct microscopic examination (DME) of KOH preparations. Good specimens are the key point that directly affects the quality of microscopic evidence and culture. The most important aspect is culturing samples on different media with or without chloramphenicol and cycloheximide and incubated at room temperature and 37°C. PCR-based no-culture methods are necessary in the cases of culture negative, morphologic identification not success, and, to prove the species identified by routine. Early treatment could save a patient's life. We start treatment at the time we have the proof of fungal infection, i.e., KOH positive. Itraconazole, fluconazole, terbinafine, amphotericin B or its liposome form, can be used alone or in combination based on the fungal species involved and the site of infection.



Scheme for diagnosis and treatment of invasive fungal infections.

State-of-the Art Lecture 2

Friday, July 15, 2011, 11:15–11:45
Meeting Room 309

SOTA2 Highlights of the APASL Single Topic on HCV in Chiba

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The diagnosis and treatment of HCV (Hepatitis C Virus) has been changing drastically for the last 20 years. Therefore, we feel the need to revise the guidelines maybe every 2 to 3 years to update the knowledge for clinical use.

The last publication on HCV guideline was 1992 by Dr. Geoff McCaugh and and myself with 17 co-authors. However after that, there is remarkable progress in understanding of the pathogenesis and natural courses of HCV infection. For example, the discovery of IL28β SNP revolutionized and changed interferon treatment indications.

Recently, the SNP analysis chronicle HCV infection was performed on large scale matters in Japanese patients, and it was revealed several significant SNPs were identified to influence on the natural courses of the hepatitis virus infection. Furthermore, the remarkable effort to develop the innovative drugs for the treatment and cure of HCV infection has been conducted for the last 10 years, and new Protease Inhibitors will be on the market shortly. These new drugs may change the paradigm of the treatment of HCV infections totally, and we are now in vision even the total cure of the diseases. Therefore, we are now in process to revise APASL guidelines to be able to catch up the incredible progress of the management on HCV infections.

Concurrent Session 4: Hepatitis C – The Key Concepts with New Therapies

Friday, July 15, 2011, 13:30–15:00
Meeting Room 310

CS4.1 Personalized medicine for HCV treatment

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Personalized therapy based on current standard of care, pegylated interferon-α (PegIFN) plus ribavirin (RBV), has become the mainstay for treatment of chronic hepatitis C virus (HCV).

Genotype-guided therapy, based on HCV genotype, recommended a 48-week regimen of PegIFN plus weight-based RBV (1000–1200 mg/d) for HCV genotype 1 or 4 (HCV-1/4); and a 24-week regimen of PegIFN plus fixed, low dose of RBV (800 mg/d) for HCV-2/3 infection. The sustained virological response (SVR, serum HCV RNA <50 IU/ml throughout 6 months of post-treatment follow-up period) rate is around 50% and 83% for HCV-1/4 patients and HCV-2/3 patients, respectively in Caucasian; and 70–75% and 85–90% in Asian patients, respectively. The optimal regimens for HCV-5 or 6 remain to be determined.

Recently, **response-guided therapy**, based on on-treatment virological responses, has become the new era of care. A shorter 24-week regimen for HCV-1 with lower baseline viral loads and a rapid virological response (RVR, serum HCV RNA <50 IU/ml at 4 weeks of treatment) and an abbreviated 16-week regimen with weight-based, standard dose of RBV for HCV-2/3 could provide equal efficacy to genotype-guided SOC. By contrast, treatment should be stopped for HCV-1 patients without an early virological response (EVR, serum HCV RNA <50 IU/ml or a 2 logs decline of HCV RNA from baseline after 12 weeks of therapy). Furthermore, EVR has been suggested to be subdivided into RVR, complete EVR (no RVR, but HCV RNA <50 IU/ml at week 12) and partial EVR (HCV RNA >2 log drop in HCV RNA but still detectable at week 12) to further improve the prediction of patients who are likely to achieve an SVR and may allow for extending treatment duration to 72 weeks, especially for those of slow responders.

More recently, the development of direct antiviral agents (DAA, formally named specifically targeted antiviral therapy for HCV, STAT-C) is ongoing. DAA has increased the number